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Atypical Cardio - Respiratory Response in a Patient with Fat Embolism - Case Report and Review of Literature

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Abstract: ***Fat embolism syndrome (FES) is a life - threatening complication in patients with orthopaedic trauma, especially long bone fractures. The diagnosis of fat embolism is made by clinical features alone, there may not be any specific diagnostic laboratory findings. FES has no specific treatment and requires only supportive care. We report the case of FES in a 36 year old male patient with Closed fracture of distal one - third shaft of Tibia and Fibula following a motor vehicular accident. The atypical haemodynamic presentation of bradycardia with hypotension presented a diagnostic challenge but it was eventually diagnosed as FES by Gurd criteria after excluding the other differential diagnoses. The patient received supportive management for the same.***

Keywords: Case report, Fat embolism, Atypical presentation

Key Messages: FES is diagnosed based on clinical features alone and requires a high degree of suspicion in patients with orthopaedic trauma; especially when it presents with atypical signs and symptoms

1. Introduction

Fat embolism syndrome (FES) is a systemic inflammatory cascade affecting multiple organ systems due to dissemination of fat emboli within the systemic circulation leading to characteristic clinical manifestations. In 1873, Bergman first described FES as a triad of confusion, dyspnoea and petechiae following long bone fractures. It is a life - threatening condition with high mortality and morbidity, with careful clinical examination being the gold standard for diagnosing FES. [1]

2. Case History

A 36 - year - old male patient with closed fracture of distal one third shaft of tibia fibula following a motor vehicular accident, was scheduled for emergency fixation of the fracture. In the operation theatre, patient was conscious, alert, comfortable with stable vitals but with a SpO_2 of 84% with no evidence of respiratory distress. The surgery was deferred and since the immediate suspicion was pulmonary embolism, a CT pulmonary angiogram was done which was normal. He was monitored in the Intensive Care Unit with oxygen on flow via facemask; SpO_2 increased to 90 - 93% and vitals were stable. But at about 16 hours the Glasgow Coma Scale suddenly worsened, and the patient developed bradycardia with hypotension not responding to intravenous fluids. The time - bound evolution of patient's clinical picture and management at each step is outlined in Table 1. Now we had to rule out a myriad of differential diagnoses using the appropriate tests which are enlisted in Tables 2, 3 and 4. The other investigations were as follows: D - dimer levels - elevated; Coagulation profile - normal; Chest X - ray - normal; CT Brain, HRCT - Thorax and CT - cervical spine - normal; COVID - 19 by RT - PCR on two occasions - negative; 2D - Echocardiography: Mild RA/RV dilatation, with Mild PAH (PASP=46), normal cardiac function; MRI Brain – not done

Finally, 24 hours after the accident, the patient developed petechiae in the axilla, conjunctiva which clinched the diagnosis of FES after all the other diagnoses were excluded.

The diagnosis of FES was made by clinical criteria proposed by Gurd and Wilson, [2] requiring two major criteria or one major criterion plus four minor criteria to make the diagnosis (table 5)

Supportive management in the form of mechanical ventilation, inotropes and blood transfusions was given. Patient was tracheostomised on day seven of intubation and then gradually weaned off the ventilator. GCS gradually improved to E4VTM3, but patient continued to have features of Central Nervous System injury. Patient had altered mental status, appeared confused, had Grade 3/5 power in bilateral upper and lower limbs with hypertonia. No seizures were noted during hospital stay. In the third week of admission, patient also developed hypertension and was started on Amlodipine. Hypertension was attributed to autonomic dysfunction in the patient secondary to cerebral injury. Patient also developed multiple episodes of febrile illness during hospital stay. Initially received antibiotics - Piperacillin - Tazobactam for a duration of 21 days and subsequently was upgraded to Cefotaxime given for a total of 14 days in view of hospital acquired pneumonia. Other supportive management in form of high protein diet by nasogastric tube, extensive limb and chest physiotherapy, mineral and vitamin supplementation and supportive management of fracture tibia and fibula with Plaster of Paris slabs was given.

Gradually, patient symptomatically improved and was eventually discharged from the hospital with residual motor and cognitive neurological deficits.

3. Discussion

Fat embolism syndrome is a rare clinical syndrome that follows an identifiable injury, which releases fat globules into the circulation resulting in pulmonary and systemic signs and symptoms. It is usually observed in patients with long bone

fractures (0.9% - 2.2%) but it has also been described in certain orthopaedic procedures, with simultaneous bilateral knee arthroplasty being one of the most frequent (0.17%)

It is a diagnosis of exclusion. Diagnosis relies on identifying risk factors, clinical signs and symptoms, laboratory results and imaging findings in an appropriate clinical setting and exclusion of other differential diagnosis. Ventilation perfusion mismatch is a hallmark of fat embolism syndrome. The arterial blood gas analysis usually has low partial pressure of oxygen, causing hypoxaemia. An increased alveolar arterial (A - a) gradient is common in fat embolism syndrome. [3] Laboratory and radiological investigations may be non - specific. The normal A - a gradient is usually less than 10 mm Hg. This can increase significantly in fat embolism syndrome because of ventilation - perfusion mismatch.

In our patient ABG revealed hypoxaemia with an A - a gradient of 215.8 mm Hg. The usual response to hypoxaemia is hyperventilation, but our patient was comfortably breathing with no apparent signs of respiratory insufficiency or haemodynamic instability. Therefore, although our first differential diagnosis was FES we also suspected pre - existing infection with COVID 19. But literature research revealed that the absence of dyspnea may not be specifically related to COVID - 19 but may occur in any patient with acute hypoxaemic respiratory failure exhibiting normal respiratory muscle function and relatively normal respiratory system mechanics. [4]

The differential diagnosis of hypotension and bradycardia is broad. There are cardiac causes such as myocardial infection with cardiogenic shock, metabolic causes such as hyperkalaemia, and neurological causes such as spinal cord injury. Patient was investigated and CT Brain along with CT Cervical Spine, electrocardiography, 2D echocardiography, serum electrolytes were done to rule out the above possibilities. Our patient had bradycardia along with hypotension, as opposed to tachycardia which is expected as a response to hypotension which presented a diagnostic challenge.

CT scan of chest may show ground glass opacities with patchy infiltration shadows but in up to 20 percent of the cases, the CT scan may be normal. [5] In our case the CT scan of thorax and brain were normal. Mortality may be as high as 30 percent, with Age> 65 years and non - orthopaedic conditions being independent risk factors for mortality. Treatment of the condition is primarily supportive, with most patients requiring respiratory support. Other supportive measures include Blood product transfusions, treatment of the orthopaedic condition. Use of Corticosteroids may be associated with reduced mortality of FES. [6]

Many studies have also shown the presence of hypoalbuminemia in fat embolism and albumin can be used for volume resuscitation. Albumin also binds harmful free fatty acids known to play a role in the pathophysiology of fat embolism and has shown decrease the incidence of fat embolism and lung injury in rat models. However, no such studies are available in human participants. Animal studies have also demonstrated benefit of using N - acetylcysteine in reducing lung injury in fat embolism syndrome. However, no such studies have been conducted in humans and its role is not established.

Anticoagulation is not commonly used as the benefit remains unproven. Other newer modalities like insertion of inferior vena caval filters prior to surgery have been suggested as means to reduce FES.

4. Conclusion

FES remains a diagnostic challenge due to its variable presentation. A high clinical index of suspicion is required, and the diagnosis is only confirmed when other pathologies with similar presentation are ruled out. Treatment is mainly supportive, and prevention is the key.

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Table 1

Time since injury	Clinical findings	Differential diagnosis	Tests done	Management
3.5 hours	Alert, oriented. Vitals stable. Injuries as described in (1)	MVA with Polytrauma	Xray long bones as described in (2)	Immobilisation of fracture site done; surgical management planned
4 hours	Alert, oriented SpO2 84 % No tachycardia	Suspected Fat embolism	CT pulmonary angiogram - normal	Face mask O2[at]5 litres/min

	No tachypnoea			
16 hours	GCS worsened to 3/15 SpO2 90 - 93% on face mask O2 Temp: 100.3 F **Bradycardia – 40 bpm** **Hypotension** not responding to volume resuscitation	1) Fat embolism 2) Spinal shock 3) Myocardial infarction with cardiogenic shock 4) Hyperkalaemia 5) COVID - 19 infection 6) Aspiration 7) Pericardial Tamponade	Haemoglobin Platelet count Serum Electrolytes RFTs Coagulation profile: PT, INR, aPTT, D - Dimer Urine - R/M Urine Fat globules Chest Xray, CT Brain + Cervical Spine, HRCT Thorax, ECG 2D – ECHO, COVID - 19 RT – PCR, (test reports mentioned in (3))	Intubation and Mechanical Ventilation Fluid resuscitation Inotropes: Dopamine
After 24 hours	GCS: E1VtM1 SpO_2: 98% (intubated on mechanical ventilation) Heart rate: 50 beats/min Blood pressure: 100/60 mmHg on inotropic support Petechiae were noticed in the axilla, conjunctiva, upper chest	FES		Supportive management was continued

Evolution of clinical picture of patient in first 24 hours of admission to Intensive care unit.

Table 2

Day of admission	1	2	3	4	7	10	14
Hb	11.5	10.8	8.9	7.4	8.9	8.7	9.13
PCV	36	35	31	25.9	29.8	29	29
Platelet count	95000	45000	50000	150000	200000	314000	430000

Haemoglobin and haematocrit - dropped on serial blood sampling

Table 3

Day of admission	1	3	5
pH	7.40	7.41	7.40
HCO3	19.1	21.5	20.4
PCO2	31.5	34.4	32.2
PaO2	140	169	152
SO2	98.2	99.5	99
BE	- 3.8	- 2.8	- 3

ABG analysis on serial blood sampling

Table 4

Day of admission	1	2	3	4	7	10	14
PT	11.6	12.4	11.6	11.6	11.9	12.4	12.2
INR	1.17	1.2	1.4	1.2	1.17	1.22	1.10
Procalcitonin	0.12	0.84	0.81		0.6	0.7	0.2
D - Dimer		7737					
CKMB	9.6						
Trop I	0.54						

Trends of Other investigations

Table 5

Gurd and Wilson Criteria for the diagnosis of Fat Embolism Syndrome
Major Criteria (one is necessary for diagnosis)
Respiratory insufficiency
Cerebral involvement
Petechial rash
Minor Criteria (Four are necessary for diagnosis)
Pyrexia (Temperature >39.4 Degrees C)
Tachycardia (Heart rate >120 beats/minute)
Retinal Changes
Jaundice
Renal Changes

Laboratory Criteria
Anaemia
Thrombocytopenia
Elevated Erythrocyte Sedimentation Rate
Fat Macroglobulinaemia (required for diagnosis)

(From Gurd AR, Wilson RI. The Fat Embolism Syndrome, J Bone Joint Surg BR 1974; 56B: 408 - 416) [2]